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PARTIAL OPTIC NERVE ATROPHY AND CENTRAL SCOTOMAS (SO-CALLED CENTRAL AMBLYOPIA), APPARENTLY DUE TO CHRONIC LEAD POISONING.*

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Illustrated.

The following cases of central amblyopia present certain clinical features of interest, and in one of them lead, so far as chemical tests are concerned, appears to have been the cause of the ocular symptoms:

Thomas Ryan, aged 35, born in England, single, a house-painter, was admitted to the medical wards of the Philadelphia Hospital on the 24th of December, 1897, on account of progressive weakness in the legs, chronic constipation, some cough with expectoration, and dim vision.

History.—The patient suffered from the usual illnesses of childhood. He denies syphilitic infection, but confesses to having had a chancroidal bubo and one attack of gonorrhea. He has been accustomed to drink spirits, and has used tobacco freely, especially by chewing, but had used neither tobacco nor liquor for two months before admission.

The man is a painter by occupation, and has followed that calling for fifteen years. His last work, however, was done five months before he entered the hospital. He has suffered at times from colic, but never typically from painter's colic, nor has he ever had wrist-drop. For the past two years he has been ailing, and for a number of years he has suffered much from persistent constipation, always requiring laxatives to secure a movement of the bowels. About November, 1897, he began to notice that his vision was failing, his attention being first directed to this symptom by his inability to read ordinary print. The failure in vision was unassociated with pain or other sign of inflammatory action.

Examination.—The patient is a somewhat emaciated, pallid man, with purplish eyelids and weak, discouraged voice. There is retraction of the left apex and harsh breathing, but no tubercle bacilli could be found in the sputa.

On admission, it is stated that examination of the urine did not reveal abnormalities, but three months later an examination by the resident, Dr. George M. Purves, gave the following result: Reaction acid; specific gravity 1017; trace of albumin with the nitro-magnesium test; no sugar; microscopically a few pale, granular casts and bladder epithelium.

There is no special disturbance of the mental faculties, except that the patient is rather slow in answering questions, this slowness being apparently due more to

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weakness and indifference than to any lack of mental qualifications. The knee-jerks are increased, and the grip of the right hand is stronger than that of the left.

Eyes.—Vision of the *right eye* 5/60; with + 4 spherical D.=2 at 25cm. The disc is nearly circular, with a scleral ring well marked all round, a sharp central excavation and faint choroidal ring outward, and some moderate choroidal disturbance in the circumference. The entire deeper layer of the disc is greenish, and considerably more than its temporal half is of a grayish-green color; superficial capillarity is preserved upon the nasal side. There are no lesions in the macula, no changes in the central circulation, and no noticeable perivasculitis.

The field of vision shows slight contraction of the form field, particularly upon the temporal side, a moderately good red field and a central scotoma, oval in form and for the most part *relative* in character, extending from 3 degrees on the nasal side to 12 degrees on the temporal side, and 3 degrees above and 7

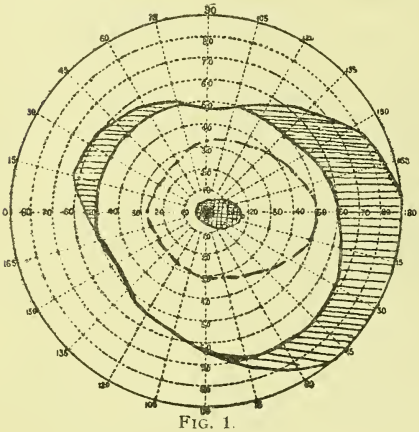


FIG. 1.

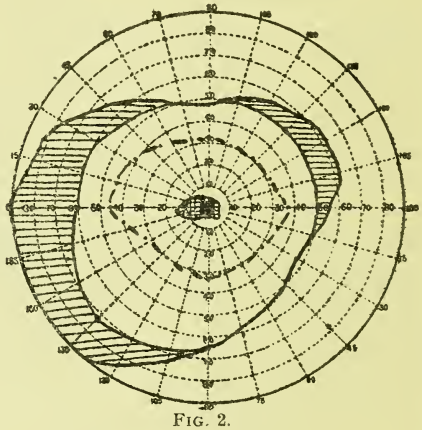


FIG. 2.

degrees below the point of fixation. Directly surrounding the fixing point and within the relative confines is a small *absolute scotoma*, about 3 degrees in diameter. (Fig. 1.)

Left Eye.—Vision 5/35; with + 4 spherical D.=2 at 25cm. The description of the fundus of the right eye already given applies also to the background on the left side, and the field of vision is very similar, containing an almost exactly similar scotoma of slightly smaller dimensions. (Fig. 2.)

The pupillary reactions are normal, and there are no palsies of the external ocular muscles.

In searching for an etiological factor to explain this case of optic nerve atrophy with central scotomas, we naturally think of toxic agents, inasmuch as the physical examinations have not given any evidence, at least any evidence at the present time, of either disseminated or fascicular disease of the cerebro-spinal axis, and syphilis seems to be excluded by the history of the case as well as this disease can ever be entirely elimi-

nated. The later examinations of the urine indicate the probable existence of chronic interstitial change in the kidneys. The patient has a phthisical history, his father having died from this disease, and he himself looks phthisical, but bacilli are not to be found in his sputa. He has taken liquor, and has chewed tobacco to excess, but has not been recently either a chewer of tobacco or a drinker of spirits, having perforce abstained from these two drugs for at least two months.

He has been a house-painter for fifteen years, and therefore it is reasonable to look for signs of lead toxemia in his system. The old sign at one time so much relied upon, namely, the blue line around the gums, was not to be demonstrated, and in this particular investigation I was aided by Dr. Frederick Packard, who has had unusual experiences in the investigation of the so-called blue line in lead poisoning.

On the 22d of February the urine was tested for lead by the method which has recently been much recommended in qualitative analyses, and which was suggested to me by Dr. Packard, namely, by means of oxylate of ammonium and a strip of magnesium, and heat in the presence of a crystal of iodine. As a result the yellow iodide of lead was deposited.

Subsequently, at my request, Dr. Alonzo Taylor attempted to make a quantitative estimation of any lead that might be found in the urine, but reported his inability to get a lead reaction, pointing out at the same time the well-known fact that the elimination of lead in the urine in cases of chronic poisoning is not constant, and that it may be lacking for days at a time without any reason being apparent. Therefore another examination was made of the urine collected for twenty-four hours, and the following is Dr. Taylor's report: "The urine contains a small quantity of lead, eight milligrammes by weight as lead. Since the quantity is minute, the natural errors of even the best method count for more than if a larger amount were concerned. The error is entirely in the direction of loss; two milligrammes may have been lost in the process. Even in well-marked cases of plumbism the urine may contain only such small quantities."

Case II.—Thomas Stevens, aged 59, born in the United States, married, a house-painter, applied for treatment in the Eye Dispensary of the Jefferson Medical College Hospital on November 26, 1897, on account of failing vision.

History.—The patient says that he has had good eyesight until eighteen months before he applied for treatment, when "vision left him in half an hour." It gradually returned, and now he sees better than at that time, but still cannot use his eyes for anything like accurate vision. The central visual acuity was $\frac{15}{cc}$ and $\frac{20}{cc}$ respectively; the ophthalmoscopic record is: both nerves grayish, retinal vessels small, central scotomas for red and green. He was referred to the Nervous Clinic for further examination; but the examinations were negative so far as central nervous disease is concerned. There was no history of syphilis or rheumatism. The knee jerks were increased.

I first examined the patient on the 12th of February, 1898, vision at that time in the right eye being 10/cc and in the left eye 20/cc. Both nerves showed general gray-white atrophy; the veins were slightly tortuous and, compared with

the arteries, large; the arteries were small, and there was well-marked, though not exaggerated, perivasculitis. Both pupils reacted normally, and there was no paresis of the external ocular muscles. The field of vision was as follows: Marked concentric contraction, especially upon the right side, very decided contraction of the red field and green blindness. In each eye there was a central scotoma extending from somewhat on the nasal side of the fixing point to about twenty degrees beyond it, in which area the appreciation of red and green was entirely lost. There was, however, no scotoma for blue, and no spots of absolute loss within the relative area.

The patient had nothing in his history which gave any good excuse for this optic nerve atrophy and perivasculitis, except exposure to lead. He smoked two cigars a day, was not a drinking man, had worked steadily at his trade, but not quite recently, had no history of painter's colic, nor had he ever suffered from wrist-drop. There were marked retraction of the gums and gingivitis. Somewhat uncertainly a blue line appeared to surround the upper incisor and left lower canine tooth. He was placed upon ascending doses of strychnine and nitro-glycerine, as he had previously taken large doses of iodide of potassium. At the end of two weeks he returned, his vision, if anything, somewhat less accurate than at the first time, namely: O.D., 10/cc.; O.S., 20/cc. Again, at the end of two weeks there was some depreciation of central vision, namely, in the right eye 4/cc, and in the left eye 15/cc. Examination of the urine failed to reveal albumin or sugar. The tests for lead, however, it was not possible to make at that time. He had carefully brushed his teeth and kept his gums in good order, so that the gingivitis had largely subsided, and still a blue line appeared, somewhat doubtfully, around the incisor and canine.

He returned to the dispensary from time to time, and at the last examination (4.6.98) the vision of the right eye was 6/cc (eccentric), and of the left eye 14/cc; ophthalmoscopically, the lesions were the same as those already recorded. There was even greater contraction of the field of vision of each eye, especially of the right, and typical central scotomas; on the right side the scotoma now included blue and white; on the left side, as before, red and green.

The faint gingival lines previously described were still visible, but no blue patches in the mucous membrane of the lip were discoverable. His urine was again submitted to an examination, which included tests for lead, with the following result: The urine contains neither albumin, sugar nor casts. There is not a trace of lead in the entire quantity submitted to examination. Lehmann's method was employed, which for qualitative work is most delicate.

The treatment was changed to ascending doses of iodide of potassium.

If we assume that the optic-nerve degeneration in these cases was due to lead—and in the first instance the chemical examinations render this assumption a reasonable one—their record seems worthy because of the somewhat unfrequent occurrence of lead amblyopia in comparison to the number of cases of chronic lead poisoning. Bearing upon this point are Dr. Frederick Packard's* interesting researches. He examined forty-eight workers in lead, the time of exposure varying from one week to

* Philadelphia Hospital Reports, III, 1896, p. 38.

twenty-six years; the average length of time of exposure among those working in lead for more than one year being nine and one-half years. Thirty-three of these men had at one time or another exhibited symptoms indicating the toxic influence of lead, but no case of amblyopia traceable to lead impregnation was found.

Lead amblyopia is also rare, in this country at least, in comparison with the whole number of ocular diseases, although it is difficult to arrive at exact figures on this subject, as some of the cases of ocular disturbance are probably hidden under the general term, toxic amblyopia. Among more than 12,000 cases of ocular disorders recorded in the Jefferson Medical College Hospital and the Philadelphia Polyclinic during the last five years, there have been only three cases of optic-nerve or retinal change attributed to lead, and two of these are doubtful.

Finally, the percentage of lead amblyopias among the total number of so-called toxic amblyopias is small. For example, Uhthoff records 204 cases of retrobulbar neuritis among 30,000 patients; of these, 138 were toxic in origin, but only one was due to lead; tobacco, alcohol, diabetes and bisulphide of carbon furnishing the remainder.

The ocular manifestations of chronic lead poisoning may manifest themselves as a transient amblyopia, due to the anesthetic effect of lead on the optic nerve and retina; as an amblyopia due to retrobulbar neuritis, which may terminate in permanent atrophy; as an optic neuritis or neuro-retinitis specifically due to lead, which may be followed by optic-nerve atrophy; and, finally, as a vasculitis and perivasculitis of the retinal vessels.*

The first of the cases reported this evening appears to belong to that group of amblyopias due to retrobulbar neuritis, which may or may not terminate in permanent atrophy. It has been argued by Stood and others that the retrobulbar neuritis of lead is analogous to the condition which lead causes in the musculo-spiral nerve, while Parisotti, on the other hand, believes that the atrophy of the papilla, which takes place without preceding neuritis, is due to changes which lead causes in the nutrient arteries of the optic nerve, changes which we know it may cause in the general retinal circulation. The character of the scotomas which have been reported in a number of cases indicates that the papillo-macular bundle is chiefly affected. This might be expected, because an abundance of clinical material indicates that this tract in the optic nerve is particularly obnoxious to the influence of toxic agents.

* Naturally, many cases of optic neuritis, neuro-retinitis, etc., seen in chronic lead poisoning, are secondary to nephritis or intracranial disorders. These cases are not now in discussion.

The second case, inasmuch as chemical examination failed to find lead in the urine, must, in spite of the presence of the slight bluish line on the gums, also somewhat uncertain in character, be relegated to the doubtful class. In this case there also appears to have been a neuritis, with special degeneration of the central fibers, the degeneration being much more widespread than in the first case, and associated with visible thickening of the coats of the blood-vessels and shrinking of their lumen.